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by

Yu. S. Alyukhin

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В в	*В в*	V, v	Т т	*Т т*	T, t
Г г	*Г г*	G, g	У у	*У у*	U, u
Д д	*Д д*	D, d	Ф ф	*Ф ф*	F, f
Е е	*Е е*	Ye, ye; E, e*	Х х	*Х х*	Kh, kh
Ж ж	*Ж ж*	Zh, zh	Ц ц	*Ц ц*	Ts, ts
З з	*З з*	Z, z	Ч ч	*Ч ч*	Ch, ch
И и	*И и*	I, i	Ш ш	*Ш ш*	Sh, sh
Й й	*Й й*	Y, y	Щ щ	*Щ щ*	Shch, shch
К к	*К к*	K, k	Ъ ъ	*Ъ ъ*	"
Л л	*Л л*	L, l	Ы ы	*Ы ы*	Y, y
М м	*М м*	M, m	Ь ь	*Ь ь*	'
Н н	*Н н*	N, n	Э э	*Э э*	E, e
О о	*О о*	O, o	Ю ю	*Ю ю*	Yu, yu
П п	*П п*	P, p	Я я	*Я я*	Ya, ya

*ye initially, after vowels, and after ъ, ь; e elsewhere.
When written as ё in Russian, transliterate as yё or ё.

RUSSIAN AND ENGLISH TRIGONOMETRIC FUNCTIONS

Russian	English	Russian	English	Russian	English
sin	sin	sh	sinh	arc sh	$\sinh^{-1}$
cos	cos	ch	cosh	arc ch	$\cosh^{-1}$
tg	tan	th	tanh	arc th	$\tanh^{-1}$
ctg	cot	cth	coth	arc cth	$\coth^{-1}$
sec	sec	sch	sech	arc sch	sech^{-1}
cosec	csc	csch	csch	arc csch	csch^{-1}

Russian	English
rot	curl
lg	log

ENVIRONMENTAL OXYGEN TENSION AND ELECTRICAL ACTIVITY OF THE BRAIN IN HYPOTHERMIA

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The question on what relation does the oxygen requirement of cells has to its actual satisfaction in the state of hypothermia has been repeatedly discussed in the literature. In the 40's a hypothesis was stated concerning the leading role of oxygen insufficiency among the physiological factors of general cooling [10, 16, 17]. In the 50's this hypothesis was placed under a doubt [5, 8, 14, 15]. It was

also shown many times that the organism withstands the hypoxic conditions in the state of hypothermia easier than at the normal body temperature. As a result, hypothermia found a broad application in the clinical practice for the prevention of hypoxic conditions. Nevertheless, a number of factors still attest in favor of the fact that hypothermia in its own right creates an oxygen insufficiency in the organism. The number of these factors includes the drop in pO_2 in the blood as a result of the shift to the left of the oxyhemoglobin dissociation curve under the effect of cooling ([7, 12] et al.), a prolonged life expectancy of animals in the state of hypothermia if the cooling is accomplished in the average atmosphere at an increased pressure [10], a progressive accumulation of excess lactate in the blood with a decrease of body temperature [9] and others. Thus, in the most recent literature one can encounter indications to the presence of oxygen difficiency during hypothermia [4, 9, 13].

We have attempted to explain how the oxygen conditions of the brain change during cooling by using the reaction of electrical activity of the brain to the change in the oxygen content in the environment. Electrical activity of the brain was selected by us as an indicator which characterized the level of functional activity of the brain during hypoxia [6] and which is connected with the oxygen requirement of the brain cells [11].

METHODOLOGY

The experiments were conducted on white rats. The rats were placed in special devices which ensured that the animal could not move its head. The temperature of the surface layers of the brain and of the rectum (Ct°) was determined by means of thermocouples. To observe the electrical activity of the cerebal cortex of large hemispheres (EKoA), electrodes were inserted in the temple bone along the sagittal suture at a distance of approximately 2 mm from the latter and 5 mm from each other. The recording of EKoA was accomplished by means of an amplifier UBP-1-02, an electron area integrator of pulses of the Kozhevnikov system [3], pulse frequency counter VSP which counts the high-frequency component of the EKoA, and a loop oscillograph N-102-T.

The cooling of animals was accomplished inside a thermal vacuum chamber with a capacity of 50 l, equipped with a coil through which cooled ethylene glycol circulated. As the air temperature in the thermal vacuum chamber dropped gradually the animal cooled at the approximately constant rate of 0.1° per 1 min. The cooling was accomplished without narcosis. Here rarefaction was created in the same thermal vacuum chamber to approximately 360 or 220 mm Hg. The

temperature and rarefaction inside the chamber was controlled automatically. The increased pressure of oxygen (3 or 6 absolute atmospheres) was created in a special high pressure chamber.

To evaluate the changes occurring in the EKoA, in the experiments with a simultaneous effect of cooling and rarefied air on the animals the EKoA was studied also when animals were cooled under the conditions of normal atmospheric pressure (hypothermic control) and under rarefied air under the conditions in which the temperature of the animals was maintained close to normal (hypoxic control). In the latter case we had to prevent the body temperature drop in rats under the effect of hypoxia. For this the thermal vacuum chamber was heated in such a way that the temperature in it was maintained at the level of approximately 30°.

The significance of the obtained results was evaluated statistically. In all 101 tests were performed on 101 animals.

RESULTS OF THE INVESTIGATIONS

In hypothermic control (Figs. 1B and 2A) the area of electrical potentials of the cerebal cortex (S-EKoA) decreased steadily in proportion to cooling. The frequency of high-frequency component

(subsequently we will call it simply frequency) of the potentials of the cerebal cortex (ν-EKoA), beginning with Ct° 36-33°, increased, achieved a maximum at Ct° 30-25°, then also decreased. The terminal in the activity of EKoA began at Ct° of 13.7±0.6° on the average (here and subsequently the mean and its standard error are indicated with a similar form of recording).

In hypoxic control with a rarefaction of 360 mm Hg (Figs. 1A and 2B, 36°) the S-EKoA increased immediately after the rarefaction began. Part of the animals could not withstand the exposure in a hypoxic medium with an artifically maintained normal temperature of the body and perished, in this case the mortality increased in proportion to the duration of the exposure. In the animals that survived the S-EKoA continued to remain, as a rule, on high level (after 2 h of exposure - at least twice that of the normal) for a duration of at least 3.5 hours. At the same time the frequency of the EKoA changed slightly. The slight (5%) statistically significant increase was observed during the 2nd and 3rd hours of exposure.

In the tests with cooling under the conditions of rarefaction at 360 mm Hg (Figs. 1B and 2B) we observed a natural suppression in the S-EKoA at Ct° 30-22° and ν-EKoA at Ct° 32-23° as compared with the levels of these parameters in a hypothermic control. At lower temperatures the significant differences between the levels of these

parameters in the hypothermic control and in the tests, as a rule, were absent. In both series the EKoA virtually ceased at the same temperature of the brain (in the experiment at 14.2±0.5° on the average). We did not observe any reliable cases of death at the hypothermic temperatures of about Ct° 12° in this series (the moment of death we identified with the moment when the heart ceased to beat, whose activity was recorded by means of an electrocardiograph).

In the hypoxia control with the rarefaction of 220 mm Hg (Figs. 3A and 4A) the sharp increase in the S-EKoA, immediately after the rarefaction, was replaced approximately by its sudden depression and death of the animals. In determining the average EKoA in this series we added the readings above the zero with the zero readings, therefore, the initial increase in the S-EKoA is absent in Fig. 3A(a). The ∨-EKoA between the beginning of rarefaction and the death of animals was below the normal level. The EKoA ceased after 22±5 min on the average after the rarefaction began.

Since the rarefaction of 220 mm Hg proved to be lethal at normal body temperature, in the tests with cooling such a rarefaction was created at the temperature of the brain of 26° (Figs. 3B and 4B). We did not observe any significant differences between the S-EKoA in this test and the S-EKoA in the hypothermic control. Initially the ∨-EKoA under the effect of rarefaction of 220 mm Hg was depressed and

became lower than the level of this parameter in the hypothermic control right up to Ct° 18°. The ν-EKoA approached the normal level as it cooled from Ct° 24° to Ct° 18° and at Ct° below 18° the difference became insignificant. The temperature at which the EKoA ceased in this series (14.5±0.3° on the average) differed insignificantly from the corresponding temperature in the hypothermic control. The EKoA ceased after 114±5 min on the average after the rarefaction began.

We did not observe any significant differences between the EKoA and the control values in the tests of high oxygen pressure, which was created at the temperature of the brain of 17° (3 atm) or 22° (6 atm).

The table shows the temperatures at which the EKoA ceases in various media.

In all experiments with cooling the rectal temperature was higher than that of the brain's surface layers by 2-3° on the average (depending on the degree of cooling and rarefaction).

DISCUSSION OF THE RESULTS

The rarefaction of up to 220 mm Hg at a normal body temperature caused a rapid depression of the EKoA down to zero after which the heart ceased to beat immediately. The rarefaction up to 360 mm Hg under the same conditions also caused death of part of the animals, although they lasted longer after the exposure began. In the animals that perished right until the moment of death, and also in those that withstood the exposure under these conditions, the EKoA was characterized by a significant increase in the area of potentials. Evidently, such an increase in S-EKoA should be examined as a sign of instability, dangerous for life state of the brain under the conditions of rarefaction. The γ-EKoA with a rarefaction of 360 mm Hg changed little in the hypoxic control. Thus, it is of little information in the evaluation of the stability of the brain under the conditions of rarefaction and for predicting the possibility of lethal outcome.

Evidently, if the reaction of the brain to the rarefaction during hypothermia would not change as compared with the reaction during a normal body temperature, the parameters of EKoA would change in response to the rarefaction during hypothermia in the same direction and to the same degree as that during the normal body temperature. This supposition we will call the hypothesis of a normal reaction to rarefaction. Since the rate of cooling in our tests was known, we had the opportunity to plot, on the graphs of the

dependences S- and)-EKoA on Ct°, that proposed dynamic of change of these parameters which should have occurred in case this hypothesis is valid. On Figs. 1B and 3B these hypothetical functions are depicted by a dashed line. These are functions, observed in actuality at normal body temperature (Figs. 1A(a, b) and 3A) and which are transformed in accordance with the fact that the level of the parameter at a particular hypothermic temperature should be compared not with the normal level of this parameter, assumed to be 100°/₀, but with a change in its level, observed at the same temperature in the hypothermic control.

In comparing such a hypothetical function with the dynamics of change actually observed in the S-EKoA during cooling in the hypothermic control and under the conditions of rarefaction of 360 mm Hg (Fig. 1B(a), we can see that the given degree of rarefaction during hypothermia not only does not elicit a dangerous increase in S-EKoA in comparison with a hypothermic control, but even entails a relative depression of this parameter which is significant in the area of Ct° 30-32°. In this case, death was not observed and the EKoA in the experiment ceases virtually at the same temperatures of the brain as those in the hypothermic control. Thus, it is possible to assume that the brain of the rats under these conditions is in a more stable state than under the same rarefaction on the background of normal body temperature. The relative depression in S- and, in

particular in ν-EKoA (Fig. 1B(b) in these experiments indicates that the reaction to rarefaction of 360 mm Hg during hypothermia still exists. Apparently, this is not the usual reaction to rarefaction, since with normal body temperature the rarefaction causes a deviation in both parameters in opposite directions. Consequently, depression in S- and in ν-EKoA during rarefaction in the cooling tests is specific for hypothermia. In this connection, it is interesting to note that approximately the same region of hypothermic temperatures (rectal temperature 34-24°) is, in the rats, the region of maximum stress of the thermoregulation mechanisms in the conditions of cooling ([1, 2] et al.). It is possible, that hypoxic depression in S- and ν-EKoA under these temperatures is analogous to the hypoxic depression of the electric activity of muscles during hypothermia ([1] et al.) and is connected with a hypoxic suppression of the chemical thermoregulation mechanisms.

An increase in stability of the brain to rarefaction during hypothermia is especially clearly demonstrated by the tests carried out at rarefaction of 220 mm Hg (Fig. 3B). It is possible to see that the animals cooled to Ct° to below 26° do not experience a rapid depression in EKoA down to zero, which one would expect in the case if the hypothesis on the normal reaction to such a rarefaction is valid. A full suppression of the EKoA ensues after a considerably longer period of time after the rarefaction begins; in this case, the

extinction temperature of the EKoA differs slightly from that observed in the hypothermic control. A significant increase in the S-EKoA was also not observed. Temporary depression in ν-EKoA at the Ct° 25-18° apparently has a mechanism which is similar to the relative depression in the ν-EKoA at higher hypothermic temperatures in response to rarefaction at 360 mm Hg. As the body temperature drops, in both these cases, the ν-EKoA relatively approaches the level of hypothermic control. Probably this is connected with a cooling depression of the thermoregulation. Apparently, with Ct° below 18° the thermoregulation is depressed to such a degree that even at the rarefaction of 220 mm Hg the ν-EKoA is virtually identical in the tests and in the hypothermic control.

The fact, confirmed by us, concerning the increased stability of the brain to the rarefaction during hypothermia is in agreement with the supposition of the fact that the hypothermia, in itself, does not cause the oxygen brain deficiency. Otherwise, one would expect not the weakening, but, on the contrary, an intensification in the effect of rarefaction of the inhaled air during hypothermia.

This conclusion is also supported by our experiments on the effect of high-pressure oxygen on the hypothermic rats. Under these conditions the pO_2 in the brain cells, probably, increases. However, with an increase in body temperature, the EKoA depresses

approximately to the same degree as in the control and ceases at close temperatures (see the table). If we are to assume that the hypothermic depression of the EKoA was caused by oxygen deficiency created in the brain as a result of general cooling, then in the tests with compressed oxygen one would expect an increase in the EKoA in comparison with the control level and the ceasation of the EKoA at lower temperatures than those in the control. Since we did not observe this, we conclude that the oxygen deficiency of the brain during hypothermia, probably, does not play a significant role in the hypothermic depression of the EKoA and, possibly, is totally absent.

CONCLUSIONS

1. The rarefaction of the inhaled air of 360 and 220 mm Hg causes a relative decrease in the integral area (with the temperatures of the brain at 30-22°) and frequency (at temperatures of the brain of 32-18°) of the biopotentials of the cerebal cortex of large hemispheres during hypothermia.

2. In the entire range of hypothermic temperatures, right up to the temperature at which the electrical activity of the cerebal cortex ceases (14°), the rat's brain, judging by its electrical activity, is more stable to the decrease in the oxygen content in the

inhaled air than at normal body temperature (which corresponds to the well known facts which state that the stability of an organism to the hypoxic conditions during hypothermia increases).

3. With the continued cooling of the rats the ceasation temperature of the electrical activity of the brain depends little or is completely independent of the oxygen tension in the environment.

4. Oxygen deficiency, probably, does not play a significant role in hypothermic depression of electrical activity of the rats brain.

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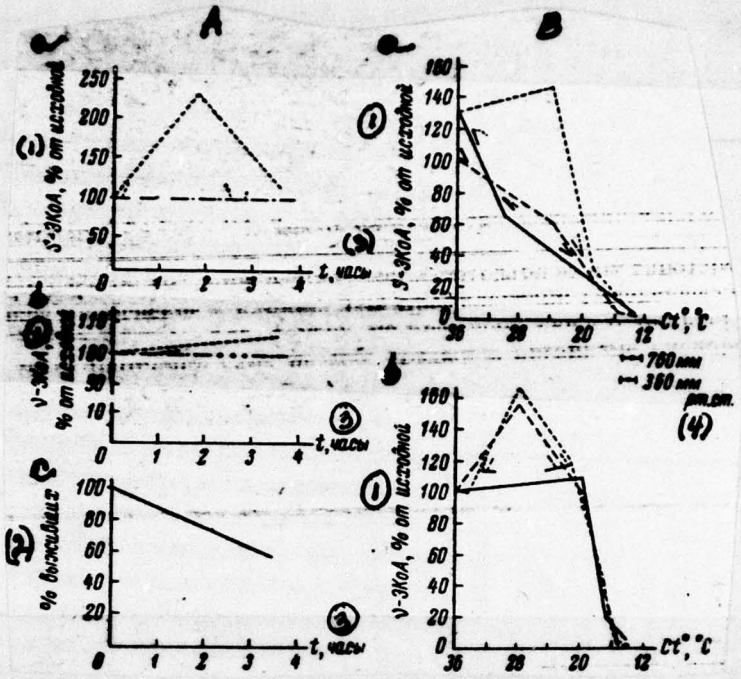

Fig. 1. Change in the area (S-EKoA) and frequency (ν-EKoA) of the electrical activity of the cerebal cortex and the survival rate of rats during the rarefaction of the inhaled air down to 360 mm Hg (schematically represented). A - at normal body temperature (hypoxic control); along the axis of the abscissas - time in hours; a - change in the S-EKoA in the survived animals; b - the same in ν-EKoA; c - percent of the survived animals. B - with a continuing general cooling; along the axis of abscissas - temperature of the brain (Ct°) in °C; horizontal segments under the abscissa axis in the B, a depicts a 95% confidence interval of medium temperatures at which the electrical activity ceases in the hypothermic control (760 mm Hg) and with a decrease in pressure down to 360 mm Hg. The solid line depicts the change in the parameters of the EKoA during a decrease in

pressure, the broken line - depicts the same in the hypothermic control, the dashed line - change into parameters, which is expected according to hypothesis under normal reaction to simulation (see the text). The apex of the angle formed by short segments with an ordinate 100% on A and one with the other on B represent points of intersection of the boundaries of the 95% confidence zones of regression with the ordinate 100% or one with the other. KEY: (1) of initial, (2) % survived, (3) hour, (4) mm Hg.

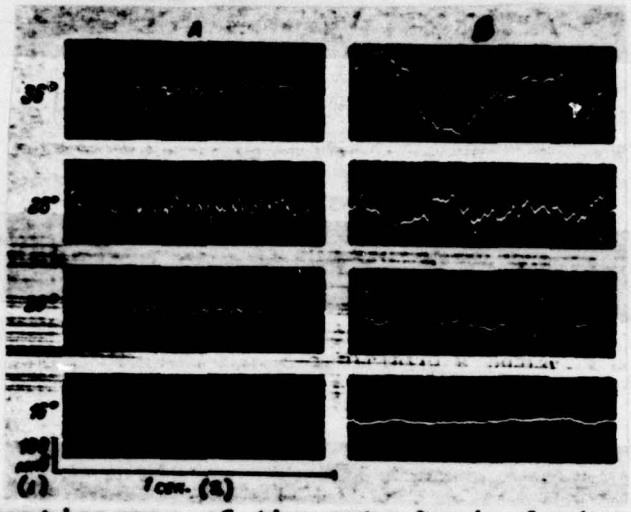

Fig. 2. Electrocorticogram of the rats brain during cooling under the conditions of normal atmospheric pressure (A) and rarefaction down to 360 mm Hg (B) as a function of the brains temperature (the numbers are on the left). KEY: (1) μV, (2) s.

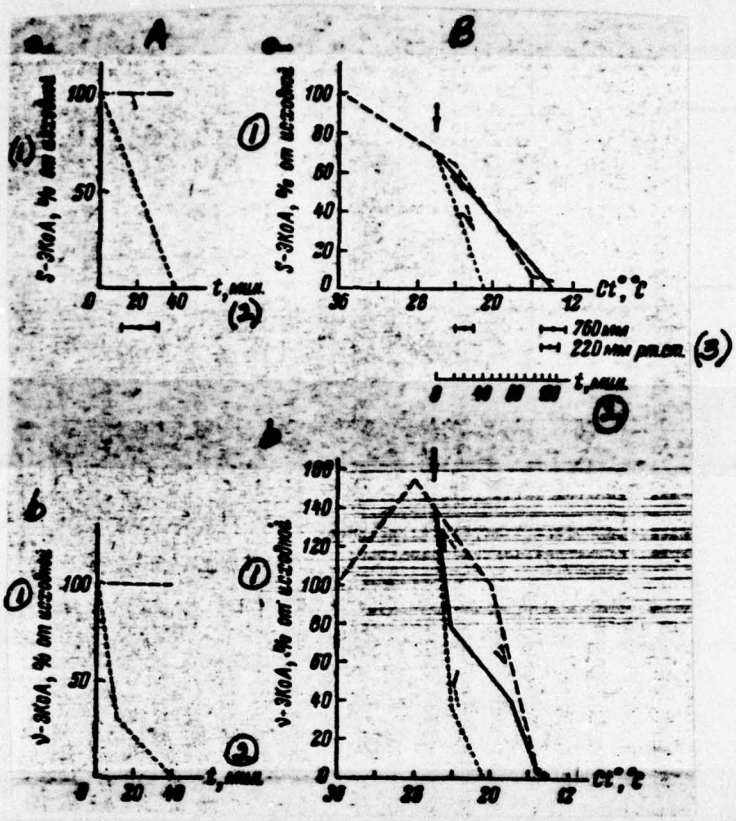

Fig. 3. Change in the electrical activity of the rats brain during a rarefaction of the inhaled air down to 220 mm Hg (drawn schematically). A - at normal body temperature (hypoxic control); the segment under the abscissas axis on a depicts 95% confidence interval of the average time at which the electrical activity ceases. B - with a continuing general cooling; arrows - start of rarefaction; additional abscissas axis in B, a - time after the rarefaction begins (t) in minutes; horizontal segments between the axes Ct° and t: segment on the left - average temperature of the brain and time at which the electrical activity ceases, which is expected according to

the hypothesis of normal reaction to rarefaction, on the right - analogous temperatures and time in the hypothermic control and in the test. The remaining designation are the same as in Fig. 1. The left boundary of the 95% confidence zone of regression in ν-EKoA in the upper portion of the graph of the hypoxic control (A, b) and regression in ν-EKoA, which is expected according to hypothesis of normal reaction to rarefaction (B, b), which is indiscernible in the scale of the drawings from the schematically drawn line of regression. KEY: (1) of initial, (2) min, (3) mm Hg.

Table. Temperatures at which the electrical activity of the rat's brain ceases during hypothermia depending on oxygen tension in the environment.

$P_{бар.}$ (1)	(2) P_{O_2} в окружающей среде (в мм рт. ст.)	n	Ct°_{f}-ЭКоА		Rt°_{f}-ЭКоА
			$\bar{x} \pm s_{\bar{x}}$ (в °С) (6A)	отличие Ct° от контроля; P (3)	
(4) Воздух (в мм рт. ст.)					
220±20	46±4	8	14.5±0.3	$0.50 > P > 0.20$	16.8±0.3
380±20	78±4	10	14.2±0.5	> 0.50	16.7±0.4
760±20	156±4	14	13.7±0.6	—	16.7±0.5
Кислород (абс. атм.) 3±0.5 (5)	2300±400	6	14.7±1.0	> 0.50	17.0±0.9

Примечание. $P_{бар.}$ — барометрическое давление; Ct°_{f} — ЭКоА — температура коры мозга, при которой прекращается ЭКоА; Rt°_{f} — ЭКоА — аналогичная ректальная температура; $\bar{x} \pm s_{\bar{x}}$ — среднее арифметическое ± оценка его стандартной ошибки; n — число наблюдений.

KEY: (1) bar, (2) environment (in mm Hg), (3) difference Ct° from control; P, (4) Air (in mm Hg), (5) Oxygen (abs. atm) 3±0.5, (6) Notation. $P_{бар.}$ - barometric pressure; $Ct^{\circ}_{f=EKoA}$ - temperature of discernible cortex at which the EKoA ceases; $Rt^{\circ}_{f=EKoA}$ - analogous rectal temperature; $\bar{x} \pm s_{\bar{x}}$ - arithmetic means ± evaluation of a standard error; n - number of observations, (6A) in.

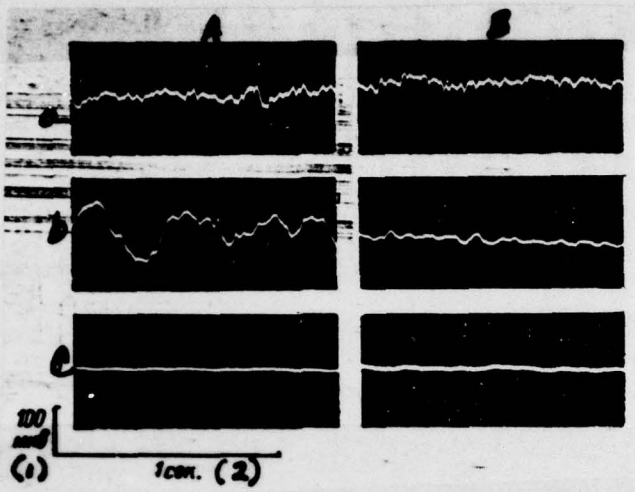

Fig. 4. Electrocorticograms of rat's brain during rarefaction to 220 mm Hg. A - at normal body temperature: a - initial; b - 7 min after rarefaction began (an increase in the area of EKoA is evident); c - 12 min after rarefaction began (complete suppression of EKoA). B - with continued cooling; a - before rarefaction, Ct° 26°; b - 50 min after rarefaction began, Ct° 20°, the arc decreases with cooling from 26 to 20° to the same degree as in the hypothermic control (comp. Figs. 2A, 28 and 20°); c - after 86 min, Ct° 16° the extinguishing activity is still noticeable. KEY: (1) μV, (2) s.

SUMMARY

Rarefaction of the inspired air to 360 or 220 mm Hg results in a relative reduction of the integral area (with brain temperatures from 30 to 22°) and frequency (with brain temperatures from 32 to 18°) of the rat cerebral cortex biopotentials in hypothermia. Over the whole range of hypothermic temperatures, up to discontinuation of the cerebral electrical activity, the brain of rats, to judge by its electrical activity, is more resistant to the fall of oxygen content in the inspired air than under conditions of normal body temperature (which is in accordance with well—known facts of the increased body resistance to hypoxic conditions in hypothermia). In an advance of cooling of rats the temperature of electrical cerebral activity discontinuation has little or no relation with environmental oxygen tension. Oxygen deficency is unlikely to be significant in hypothermic depression of electrical cerebral activity in rats.

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